

# Editorial

## Prognosis in occupational asthma

While the clinical picture of occupational asthma and methods of establishing the diagnosis are reasonably well known, the prognosis is less well documented. As asthma is a reversible airways disease, removal from the offending agent might be expected to lead to complete recovery. Unfortunately this does not always occur.

There are now several reports of follow up studies of patients with occupational asthma. In 1975 Adams<sup>1</sup> found a significant excess of respiratory symptoms in 46 patients with toluene diisocyanate (TDI) induced asthma who had been removed from exposure for from two to 11 years. Moller and co-workers<sup>2</sup> reported that seven out of 12 patients with TDI asthma had persistent asthma even though they had been removed from exposure for a mean period of 1.9 years; these patients were shown to have retained their TDI "sensitivity" by bronchial challenge testing. Paggiaro *et al*<sup>3</sup> studied 27 patients with TDI induced asthma proved by bronchoprovocation tests two years after their first examination. Eight out of 12 patients who had left the industry complained of persistent dyspnoea and wheeze and most of them had bronchial hyperreactivity as demonstrated by a methacholine challenge test. Continuation of exposure in 15 workers led to further deterioration of airflow obstruction and increase in bronchial reactivity.

Chan-Yeung and coworkers<sup>4</sup> in a follow up study of 75 individuals with asthma due to Western red cedar showed that only half recovered completely after removal from exposure. The remaining half continued to have recurrent attacks of asthma after a mean period of three years (range 1-9 years) without exposure. The severity of symptoms varied considerably from occasional attacks of dyspnoea relieved by the use of aerosol bronchodilators to persistent chronic asthma requiring systemic corticosteroids and other regular medication. Among patients with occupational asthma caused by colophony fumes Burge<sup>5</sup> reported similar findings; only two of the 20 affected workers who had left exposure were symptom free on follow up. He has, how-

ever, pointed out that colophony and pine products are widespread in the home, so that persistent symptoms may have been caused by domestic exposure.

Hudson *et al*<sup>6</sup> carried out a follow up study of patients with occupational asthma due to a variety of agents, including small and large molecular compounds. Of the 31 patients with asthma due to crab processing, 19 still had symptoms after being away from work for more than 12 months. Of the 32 workers with asthma due to a variety of agents such as isocyanate, red cedar, other wood dust, flour, antibiotics, etc, only two had recovered completely after a mean period of 24 months without exposure.

These studies show that many of the patients with occupational asthma do not recover completely after the cessation of exposure even though their condition is frequently improved. The persistence of symptoms is accompanied by the presence of non-specific bronchial hyperreactivity to methacholine or histamine.<sup>2-6</sup> As these people did not have asthma before they entered the industry we may reasonably assume that their symptoms are the result of occupational exposure. Somehow exposure to the offending agents alters the reactivity of the airways in these individuals.

We might argue that workers with occupational asthma were all going to develop late onset asthma and that occupational exposure merely unmasks the predisposition. There are, however, several points against such an argument. Firstly, in industries where occupational asthma is documented the prevalence of asthma is usually higher than expected. In British Columbia the prevalence of asthma (on the basis of a questionnaire) among red cedar sawmill workers is 10.4% which is significantly higher than the prevalence of asthma found in office workers (4.3%)<sup>7</sup>. In some groups of workers exposed to platinum salts and proteolytic enzymes as many as half have developed asthma.<sup>8,9</sup> Secondly, in patients who recovered from occupational asthma, non-specific bronchial hyperreactivity returned towards normal,<sup>4,5</sup> indicating that those sensitised acquired a disease from their job. Thirdly, among "intrinsic asthmatics" Brostoff<sup>9</sup> found an excess of homozygotes for BW6 on the HLA-B locus, but such an increase was not found in patients with occupational asthma induced by exposure to colophony fumes,<sup>5</sup> suggesting at least that those with occupational asthma do not have the same genetic predisposition as "intrinsic asthmatics."

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What factors affect the prognosis? In their follow up study of 75 patients with proved red cedar asthma, Chan-Yeung and coworkers<sup>4</sup> considered various factors, such as duration of exposure before the onset of symptoms, duration of symptoms before diagnosis, age, race, smoking, atopy, types of asthmatic reaction induced by inhalation challenge, pulmonary function, and non-specific bronchial reactivity at the time of diagnosis. They found that those with persistent asthma had a significantly longer duration of symptoms before diagnosis, poorer lung function, and a more severe degree of non-specific bronchial hyperreactivity at the time of diagnosis than those who recovered. In their follow up study of patients with occupational asthma due to a variety of agents, Hudson and coworkers<sup>6</sup> found similar prognostic factors. The findings of these two studies suggest that those with asthma persisting after cessation of exposure were diagnosed late and had more severe disease at the time of diagnosis than those who recovered. Moreover, continuous exposure to TDI in sensitised patients has been shown to lead to further deterioration in lung function and increase in non-specific bronchial reactivity.<sup>3</sup> It is therefore very important that patients with occupational asthma should be diagnosed early and removed from exposure as soon as possible.

Unfortunately most affected workers fail to find alternative employment. The employer may attempt to relocate the worker to another area of the plant with no or little exposure, but such "lateral bumping" is not allowed by some labour unions and the affected worker may have to wait until such a vacancy occurs. A transfer may bring a cut in pay and loss of seniority. Before 1981, when the economy was buoyant in British Columbia, only one third of patients with red cedar asthma continued to work in the same job after the diagnosis was made;<sup>4</sup> with worsening in the economy after 1981, however, most patients now remain in the same job. What can be done for these unfortunate affected individuals to prevent progress of the disease?

The usual recommendations are the use of respirators and appropriate medication. The dust masks are, however, often ineffective because they are ill fitting; and workers' compliance is low when they are given heavy respirators. The use of treatment such as sodium cromoglycate,  $\beta$  adrenergic stimulants, or beclomethasone dipropionate aerosol may bring useful suppression of the asthmatic symptoms but there is so far no information on whether this prevents progression of damage to the airways.

Employers should be responsible for improving the working environment using modern engineering techniques. This is important in preventing sensitisa-

tion and also making it possible for affected workers to remain in the same jobs. Consideration should be given to changes in formulation of the product whenever possible. For example, in the detergent enzyme industry encapsulation of the proteolytic enzyme portion of the product reduced the exposure of the workers. The institution of safety measures concerning handling procedures, the avoidance of spills, the promotion of good housekeeping, and the education of workers about these measures are all important. The Workers' Compensation Boards or equivalent independent authorities with responsibility for environmental health should be required to ensure that the working environment is safe for the workers by monitoring the level of exposure at regular intervals. In addition, careful medical follow up of affected individuals who are allowed to continue their exposure seems essential.

The issue of compensation of patients with occupational asthma has been addressed in detail in an earlier editorial.<sup>10</sup> In Britain compensation is available only to workers who are occupationally exposed to one of seven specific groups of "prescribed" agents. These are: platinum salts, isocyanates, epoxy resins, colophony fumes, proteolytic enzymes, laboratory animals and insects, and grain (or flour) dust. They are all potent sensitisers.

In Canada the compensation system differs from province to province. In British Columbia the diagnosis of occupational asthma can be based on the history if known sensitising agents are found in the work place and the affected individual does not have pre-existing asthma. Specific bronchial challenge tests are required only if the history is atypical or if known sensitising agents are absent in the work place. Once the diagnosis of occupational asthma is established there is no difficulty in having the patient's claim accepted by the Workers' Compensation Board and short term compensation granted.

As asthmatic symptoms persist in many affected individuals after their exposure has ceased, the question of compensation for permanent disability arises. The British Columbia Workers' Compensation Board has considerable difficulty with this problem as there are no guidelines for the evaluation of permanent impairment or disability for patients with asthma. The Board uses the guidelines published by the American Medical Association in 1977 for the respiratory system.<sup>11</sup> According to these, the affected person is not considered disabled if his or her lung function ( $FEV_1$  or forced vital capacity) is within 95% confidence limits of normal. Individuals with asthma may, however, have normal lung function, a normal chest radiograph, and normal arterial oxygen saturation while taking medication. Even if no regular treatment is taken the symptoms may be

intermittent and lung function may be normal on one day and abnormal on another. Nevertheless, these individuals have increased non-specific bronchial hyperreactivity, which may make them unable to work in environments where other pollutants act as irritants even though the specific sensitising agent is absent. Clearly the above guidelines using lung function tests are inadequate for disability assessment in asthma.

Patients with asthma have evidence of non-specific bronchial hyperreactivity even during remissions<sup>12</sup> and, furthermore, the degree of non-specific bronchial hyperreactivity is closely correlated with the severity of asthma.<sup>13</sup> In future guidelines on respiratory impairment, in addition to the use of lung function measurements, there would be much to recommend inclusion of an assessment of bronchial reactivity. Such a recommendation might require a pre-employment methacholine or histamine challenge test to determine initial bronchial reactivity.

It has been suggested that bronchial hyperreactivity is a predisposing factor in the development of occupational asthma but there is still no published prospective study of pre-employment histamine or methacholine reactivity in workers who subsequently have regular medical examinations and periodic histamine or methacholine challenge tests. Such a study would provide answers to several important questions. Is bronchial hyperreactivity a predisposing factor? If it is, should hyperreactive individuals be excluded from entering the industry? If it is not, does development of bronchial hyperreactivity precede the development of clinical symptoms? How stable is the response to methacholine or histamine challenge test and how frequently should these tests be performed?

With increased recognition of the disease and with an increased rate of introduction of new substances into industries, occupational asthma may replace pneumoconiosis as the most common occupational disease of the lung. Further research will improve our understanding of the pathogenetic mechanisms and predisposing factors of occupational asthma and it should also allow society to deal fairly with the affected individuals.

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